Harris
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for details.

=> e endothelin b receptor/cn

E1            1     ENDOTHELIN 3-LIKE PEPTIDE (HUMAN REDUCED)/CN
E2            1     ENDOTHELIN 3-LIKE PEPTIDE (MOUSE REDUCED)/CN
E3            0 --> ENDOTHELIN B RECEPTOR/CN
E4            1     ENDOTHELIN C-TERMINAL HEXAPEPTIDE/CN
E5            1     ENDOTHELIN DEGRADATION ENZYME/CN
E6            1     ENDOTHELIN ETA RECEPTOR (PIG)/CN
E7            1     ENDOTHELIN ETB RECEPTOR (SWINE SVR ISOFORM)/CN
E8            1     ENDOTHELIN HOMOLOG (HUMAN CLONE DNA55800-1263)/CN
E9            1     ENDOTHELIN I/CN
E10           1     ENDOTHELIN III/CN
E11           1     ENDOTHELIN PRECURSOR-PROCESSING PROTEINASE/CN
E12           1     ENDOTHELIN PRECURSOR-PROCESSING PROTEINASE (GUINEA PIG)/CN

=> e endothelin/cn 5

E1            1     ENDOTHELIAL-MONOCYTE ACTIVATING POLYPEPTIDE II (MOUSE CELL
L
                    INE METH A)/CN
E2            1     ENDOTHELIAL-MONOCYTE ACTIVATING PROTEIN III (HUMAN)/CN
E3            1 --> ENDOTHELIN/CN
E4            1     ENDOTHELIN (MOUSE REDUCED)/CN
E5            1     ENDOTHELIN (MOUSE REDUCED), BIG/CN

=> s e3

L1            1 ENDOTHELIN/CN

=> fil medl,caplus,biosis,embase,wpids,jicst

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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                                                        ENTRY     SESSION
FULL ESTIMATED COST                                      4.11      506.06

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                      0.00      -7.64

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FILE 'CAPLUS' ENTERED AT 14:35:53 ON 10 JAN 2001
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=> s (endothelin b receptor or etb or endothelin receptors type b) and
(cancer or melanoma) and (protstate or colon or ovarian or mammar?)

L2            3 FILE MEDLINE
L3           10 FILE CAPLUS
L4            5 FILE BIOSIS
L5            2 FILE EMBASE
L6            1 FILE WPIDS
L7            0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L8           21 (ENDOTHELIN B RECEPTOR OR ETB OR ENDOTHELIN RECEPTORS TYPE B)
                AND (CANCER OR MELANOMA) AND (PROTSTATE OR COLON OR OVARIAN OR
                MAMMAR?)

=> s (antisense or ribozyme or endothelin 1) and l8

L9            3 FILE MEDLINE
L10           7 FILE CAPLUS
L11           4 FILE BIOSIS
L12           1 FILE EMBASE
L13           1 FILE WPIDS
L14           0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L15          16 (ANTISENSE OR RIBOZYME OR ENDOTHELIN 1) AND L8

=> dup rem l15

PROCESSING COMPLETED FOR L15
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L16 10 DUP REM L15 (6 DUPLICATES REMOVED)

=> d 1-10 cbib abs

L16 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 1
2000:790734 Document No. 133:329575 **Cancer** treatment with endothelin receptor antagonists. Schneider, Robert J.; Jamal, Sumayah (New York University, USA). PCT Int. Appl. WO 2000067024 A1 20001109, 64 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US11990 20000503. PRIORITY: US 1999-305084 19990504.

AB The present invention relates to therapeutic protocols and pharmaceutical compns. designed to treat and prevent **cancer**. More specifically, the present invention relates to a novel method of treating **cancer** using antagonists to the **endothelin B receptor** (**ETB**) or inactive mimic forms of **endothelin-1**. The pharmaceutical compns. of the invention are capable of selectively inhibiting the early events assocd. with the development of **cancer**. The present invention further relates to screening assays to identify compds. which inhibit **ETB** activation.

L16 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2001 ACS
2000:133697 Document No. 132:203144 Low-adenosine **antisense** oligonucleotide agents, compositions, kits and treatments for respiratory disorders. Nyce, Jonathan W. (East Carolina University, USA). PCT Int. Appl. WO 2000009525 A2 20000224, 1343 pp. DESIGNATED STATES: W: AU, CA, CN, MX, RU, US; RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1999-US17712 19990803. PRIORITY: US 1998-95212 19980803.

AB A compn. comprises a nucleic acid comprising an oligo **antisense** to a target such as polypeptide(s) assocd. with an ailment afflicting lung airways, genes and mRNAs encoding them, genomic and mRNA flanking regions, intron and exon borders and all regulatory and functionally related segments of the genes and mRNAs encoding the polypeptides, their salts and mixts. Various formulations contain a requisite carrier, and optionally other additives and biol. active agents. The agent of the invention may be prepd. by selecting a target gene(s), genomic flanking region(s), RNA(s) and/or polypeptide(s) assocd. with a disease(s) or condition(s) afflicting lung airways, obtaining the sequence of the mRNA(s) corresponding to the target gene(s) and/or genomic flanking region(s), and/or RNAs encoding the target polypeptide(s), selecting at least one segment of the mRNA which may be up to 60% free of thymidine (T) and synthesizing one or more **antisense** oligonucleotide(s) to the mRNA segments which are free of adenosine (A) by substituting a universal base for A when present in the oligonucleotide. The agent may be prepd.

by selection of target nucleic acid sequences with GC running stretches, which have low T content, and by optionally replacing A in the **antisense** oligonucleotides with a universal base. The agent, compn. and formulations are used for prophylactic, preventive and therapeutic treatment of ailments assocd. with impaired respiration, allergy(ies) and/or inflammation, such as pulmonary vasoconstriction, inflammation, allergies, asthma, impeded respiration, lung pain, cystic fibrosis, bronchoconstriction, pulmonary hypertension and bronchoconstriction, chronic bronchitis, emphysema, chronic obstructive pulmonary disease (COPD), acute respiratory distress syndrome (ARDS), ischemic conditions including ischemia itself, and **cancers** such as leukemias, lymphomas, carcinomas, and the like, e.g. **colon cancer**, breast **cancer**, pancreatic **cancer**, lung **cancer**, hepatocellular carcinoma, kidney **cancer**, **melanoma**, hepatic metastasis, etc., as well as all types of **cancers** with may metastasize or have metastasized to the lung(s), including breast and prostate **cancer**. The present treatment is suitable for administration in combination with other treatments, e.g. before, during and after other treatments, including radiation, chemotherapy, antibody therapy and surgery, among others. The present agent is effectively administered preventatively, prophylactically or therapeutically by itself for conditions without known therapies, or as a substitute for, or in conjunction with, other therapies exhibiting undesirable side effects. The treatment of this invention may be administered directly into the respiratory system of a subject, so that the agent has direct access to the airways and the lungs. The invention is exemplified with specificity and pharmacokinetic studies using phosphorothioated **antisense** oligonucleotides targeted to the adenosine receptors A1, A2a, A2b, and A3.

L16 ANSWER 3 OF 10 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
2000438062 EMBASE Modulation of human **colon** tumor-stromal interactions by the endothelin system. Egidy G.; Juillerat-Jeanneret L.; Jeannin J.-F.; Korth P.; Bosman F.T.; Pinet F.. Dr. F. Pinet, INSERM Unit 36, College de France, 3 rue d'Ulm, 75005 Paris, France. florence.pinet@college-de-france.fr. American Journal of Pathology 157/6 (1863-1874) 2000.
Refs: 41.
ISSN: 0002-9440. CODEN: AJPAA4. Pub. Country: United States. Language: English. Summary Language: English.

AB Tumor neovascularization is considered to be a critical step in the development of a malignant tumor. Endothelin (ET)-1 is a powerful vasoconstrictor and mitogenic peptide that is produced by many **cancer** cell lines. The cellular distribution of the ET components was evaluated in human **colon** tumors and compared to normal **colon**. There was more of the ET components (preproET-1, endothelin-converting enzyme-1, and ETA and **ETB** receptors) in adenomas and adenocarcinomas than in the normal **colon**. There was overproduction of preproET-1 and endothelin-converting enzyme-1 in carcinoma cells and stromal vessels, suggesting that they are a local source of ET-1. ETA receptors were present in stromal myofibroblasts of neoplastic tissue, and there were large amounts of **ETB** receptors in the endothelium and myofibroblasts. There was also a redistribution of .alpha.-smooth muscle actin-positive cells in the vascular structures of tumors. An experimental rat model of induced **colon cancer** treated for 30 days with bosentan, a mixed antagonist of

both ET receptors, confirmed the morphological changes observed during the tumor vascularization. Our data suggest that ET-1 and its receptor play a role in **colon cancer** progression, with ET-1 functioning as a negative modulator of the stromal response.

L16 ANSWER 4 OF 10 MEDLINE DUPLICATE 2
2000211278 Document Number: 20211278. Studies on the expression of endothelin, its receptor subtypes, and converting enzymes in lung **cancer** and in human bronchial epithelium. Ahmed S I; Thompson J; Coulson J M; Woll P J. (Cancer Research Campaign, Academic Department of Clinical Oncology, University of Nottingham, Nottingham City Hospital, United Kingdom.) AMERICAN JOURNAL OF RESPIRATORY CELL AND MOLECULAR BIOLOGY, (2000 Apr) 22 (4) 422-31. Journal code: AOB. ISSN: 1044-1549. Pub. country: United States. Language: English.
AB Lung **cancer**, particularly small cell lung **cancer** (SCLC), is characterized by production of numerous peptides and their resulting clinical syndromes. Such peptides can act as autocrine growth factors for these tumors. In this study, we investigated the role of endothelin (ET)-1 in lung **cancer**. Using reverse transcription/polymerase chain reaction (RT-PCR), enzyme-linked immunosorbent assay, and immunocytochemistry, we screened a panel of lung **cancer** cell lines for ET-1, its receptors, and endothelin converting enzyme-1 (ECE-1), which generates the active form of ET-1. ET-1 messenger RNA was expressed in five of seven SCLC, four of four non-small cell lung **cancer** (NSCLC), and human bronchial epithelial (HBE) cells. The intracellular isoform of ECE-1, important in processing ET-1 if an autocrine growth loop is to function, was downregulated in the lung **cancer** cell lines as compared with expression of the extracellular isoform. Endothelin A receptor (ETAR), which mediates the mitogenic effects of ET-1 in prostate and **ovarian cancer**, was upregulated in HBE cells compared with expression in three of seven SCLC and two of four NSCLC cell lines. **Endothelin B receptor** (ETBR) was more widespread, being expressed in seven of seven SCLC, four of four NSCLC, and the HBE cells. We used flow cytometry to measure mobilization of intracellular calcium as a functional assay for the ETAR. These data concurred with the RT-PCR results, indicating that the ETAR was downregulated or was involved in an alternative signal transduction pathway in lung **cancer**, and no evidence of functional receptor mediating an autocrine growth loop was found. From our study, the data do not support the putative functional autocrine growth role of ET-1 in lung **cancer**. We propose instead that ET-1 may act as a paracrine growth factor for surrounding epithelial and endothelial cells via alternative pathways, promoting angiogenesis and stromal growth.

L16 ANSWER 5 OF 10 BIOSIS COPYRIGHT 2001 BIOSIS
2000:164968 Document No.: PREV200000164968. Augmented expression of **endothelin-1**, endothelin-3 and the **endothelin-B receptor** in breast carcinoma. Alanen, K.; Deng, D.-X.; Chakrabarti, S. (1). (1) Department of Pathology, University of Western Ontario, London Health Sciences Centre, London, ON, N6A 5A5 Canada.

Histopathology (Oxford)., (Feb., 2000) Vol. 36, No. 2, pp. 161-167. ISSN: 0309-0167. Language: English. Summary Language: English.

AB Aims: Endothelins (ETs) are peptides expressed in many tumours which may stimulate angiogenesis and desmoplasia. Because ETs have not been extensively studied **mammary** neoplasia, we assessed ET protein and mRNA expression and receptor mRNA expression in normal and neoplastic breast tissues. Methods and results: Tissues from five normal breasts, six fibroadenomas, seven ductal carcinomas in situ (DCIS) and 25 invasive carcinomas were stained with anti-ET-1 and anti-ET-3 antibodies and analysed using a grading system. ET-1, ET-3, ETA and **ETB** mRNA expression was assessed by quantitative RT-PCR from eight carcinomas and five normals. Weak staining for ET-1 and ET-3 was detected in all normals. Moderate to strong staining was seen in 72% and 64% of carcinomas for ET-1 and ET-3, respectively. Most fibroadenomas showed weak positivity for ET-1 (83%) and ET-3 (67%). ET-1 and ET-3 mRNA levels were upregulated in carcinomas compared with normal breast. No ETA mRNA was not detected in any tissue. **ETB** mRNA was detected in normal breast and was increased in carcinomas. Conclusion: These results suggest that the ET system is altered in breast carcinomas and this may be of importance in the progression from in-situ to invasive carcinoma.

L16 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2001 ACS

1999:722912 Document No. 131:317804 Methods for treatment of pain by inhibiting **endothelin-1** action. Davar, Gudarz (USA). PCT Int. Appl. WO 9956761 A1 19991111, 39 pp. DESIGNATED STATES: W: AU, CA, JP; RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1999-US9732 19990504. PRIORITY: US 1998-72428 19980504.

AB A method of detg. whether a compd. alleviates nerve pain mediated by **endothelin-1** (ET-1) involves (i) detg. whether the compd. has the ability to inhibit a ET-1 action and then (ii) detg. whether the compd. reduces nerve pain by testing the compd. in human patients suffering from pain mediated by the ET-1 action. The invention also includes a method of detg. whether a compd. alleviates pain caused by nerve injury in human patients by detg. the compd. ability to inhibit an inflammatory leukocyte response. ET-1 (40-800 .mu.M) applied to rat sciatic nerve in vivo induced direct effect on sensory neurons and pain behavior via a mechanism independent of vasoconstriction of sciatic nerve microvessels. ET-1-induced pain behavior is mediated by ATA subtype of receptor on neurons, as evidenced by using ETA and **ETB** receptor antagonists, BQ-123 and BQ-788, resp. Therefore, the inhibition of ET-1's vasoconstriction-independent mechanism of causing pain is an effective pain treatment, esp. under conditions where ET-1 levels are elevated in a patient, such as metastatic prostate **cancer**. Furthermore, given that ET-1 acts directly on the sensory neuron ETA receptor, the ETA receptor is an important therapeutic target.

L16 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2001 ACS

1999:654633 Document No. 132:91570 Endothelins and their receptors in cirrhotic and neoplastic livers of Canadian and Chinese populations. Cai,

L.; Wang, G. J.; Mukherjee, K.; Xu, Z. L.; Khalil, M.; Cherian, M. G.; Chakrabarti, S. (Department of Pathology, The University of Western Ontario, London, ON, N6A 5C1, Can.). Anticancer Res., 19(3B), 2243-2247 (English) 1999. CODEN: ANTRD4. ISSN: 0250-7005. Publisher: International Institute of Anticancer Research.

AB Background: Endothelins (ETs) are 21 amino acid peptides with widespread tissue distribution and functions. In this study, we retrospectively investigated immunoreactive ET-1, ET-3 as well as ET receptors by ligand binding and autoradiog. in hepatic cirrhosis and neoplasms. Formalin fixed paraffin embedded tissues from 30 hepatocellular carcinomas (HCC), 4 fibrolamellar carcinomas (FLC), and 7 liver metastatic adenocarcinomas (Ad) from **colon** were collected from the Pathol. Department of London Health Science Center. Adjacent cirrhotic livers were obtained from 17 cases and adjacent normal liver was present in 12 cases. In addn., 15 HCCs, 6 cirrhotic and 8 normal livers were obtained from Normal Bethune University for Medical Sciences in China. The slides were stained for ET-1 and ET-3 with a polyclonal antibody and scored. Autoradiog. localization of ET-receptors with 125I-ET-1 was carried out in some of the cases. In the normal liver, hepatocytes, biliáry epithelium, vascular endothelium and smooth muscle cells were pos. for both ET-1 and ET-3. Higher immunoreactivity for ET-1 and ET-3 was seen in cirrhosis. HCCs showed variation in immunoreactivity, with overall scoring not different from normal livers. FLCs showed consistent higher immunoreactivity for both ET-1 and ET-3, while in Ads the immunoreactivity was decreased. Increased ET-receptors, representing both ETA and **ETB** subtypes were seen in both cirrhosis and in HCC. Alterations in both ETs and their receptors were found in cirrhosis and neoplastic liver diseases.

L16 ANSWER 8 OF 10 MEDLINE DUPLICATE 3

2000123001 Document Number: 20123001. Paracrine regulation of **ovarian cancer** by endothelin. Moraitis S; Miller W R; Smyth J F; Langdon S P. (Imperial Cancer Research Fund Medical Oncology Unit, Western General Hospital, Edinburgh, U.K.) EUROPEAN JOURNAL OF CANCER, (1999 Sep) 35 (9) 1381-7. Journal code: ARV. ISSN: 0959-8049. Pub. country: ENGLAND: United Kingdom. Language: English.

AB Previous studies have demonstrated that endothelin (ET) isoforms (ET-1, ET-2 and ET-3) can act in an autocrine manner in **ovarian cancer** while in breast **cancer** their role has been proposed to be that of a paracrine mitogen. To explore the possibility that endothelin isoforms might function not only as autocrine regulators but also as paracrine mitogens in **ovarian cancers**, we investigated their effects on the growth of **ovarian** fibroblasts derived from **ovarian** carcinomas, the interaction between **ovarian** carcinoma and fibroblast cells and the location of the isoform expression in primary **ovarian** tumours. ET-1, ET-2 and ET-3 stimulated the growth of three **ovarian** fibroblast cell lines at concentrations ranging from 10(-12) M to 10(-7) M. Inhibition of 125I-ET binding by the ETA receptor antagonist BQ123 and the **ETB** receptor antagonist BQ788 suggested the presence of both types of ET receptors in fibroblast cells. In the absence of ET-1, neither BQ 123 nor BQ 788 inhibited growth. However, both antagonists inhibited ET-1 stimulated growth suggesting the involvement of both receptor types in

ET-1 growth regulation. In contrast to carcinoma cells which secrete measurable levels of ET-1, fibroblast cell lines did not secrete detectable protein. Co-culture experiments (using porous membrane insert wells) of fibroblasts with carcinoma cells demonstrated that growth of both populations of cells was increased compared with either grown in isolation. In this system, growth of the fibroblast cell line was partially inhibited by both BQ123 and BQ788, whilst growth of the PE014 carcinoma cell line was inhibited by only BQ123. RT-PCR measurements detected the presence of the ETA receptor subtype in 10/10 primary **ovarian cancers** but the presence of **ETB** receptor in only 6/10 **cancers**. Using specific antibodies, ET-1 was found in 11/15, ET-2 in 5 of 7 and ET-3 in 5/7 primary **ovarian cancers** predominantly in the epithelial cells but with some stromal expression. These data indicate that the ET isoforms may stimulate growth of the fibroblast population within an **ovarian cancer** in addition to stimulating the epithelial cells and since the ETs are expressed in the majority of **ovarian cancers**, this paracrine effect may contribute to the overall growth of the tumour.

L16 ANSWER 9 OF 10 BIOSIS COPYRIGHT 2001 BIOSIS
1999:441523 Document No.: PREV199900441523. Endothelin receptor expression in colorectal **cancer**. Ali, H. (1); Loizidou, M. (1); Dashwood, M.; Savage, F. (1); Boulos, P. B. (1); Taylor, I. (1). (1) Department of Surgery, Royal Free and University College Medical School, London UK. British Journal of Cancer, (July, 1999) Vol. 80, No. SUPPL. 2, pp. 91. Meeting Info.: Joint Meeting of the British Association for Cancer Research, the British Oncological Association, the Association of Cancer Physicians and the Royal College of Radiologists Edinburgh, Scotland, UK July 11-14, 1999 ISSN: 0007-0920. Language: English.

L16 ANSWER 10 OF 10 MEDLINE DUPLICATE 4
97419945 Document Number: 97419945. Endothelin expression and responsiveness in human **ovarian** carcinoma cell lines. Moraitis S; Langdon S P; Miller W R. (Imperial Cancer Research Fund Medical Oncology Unit, Western General Hospital, Edinburgh, U.K.) EUROPEAN JOURNAL OF CANCER, (1997 Apr) 33 (4) 661-8. Journal code: ARV. ISSN: 0959-8049. Pub. country: ENGLAND: United Kingdom. Language: English.
AB To elucidate the potential role of endothelins (ETs) as growth regulators in **ovarian** carcinoma cells in culture, expression of endothelins and their receptors were measured in two **ovarian cancer** cell lines (PEO4 and PEO14), together with the effect of the exogenous addition of endothelins on the growth of these cell lines in vitro. RT-PCR analysis of mRNA prepared from PEO4 and PEO14 indicated the presence of ET-1 and ET-3 mRNA. Immunoreactive ET-1-like peptide was found in media from cultures of both PEO4 (1.7 +/- 0.4 fmol/10(6) cells/72 h) and PEO14 (20.2 +/- 6.8 fmol/10(6) cells/72 h) cell lines. Radioligand binding studies using 125I-ET-1 and membrane fractions were consistent with PEO4 cells having two receptor sites of either high affinity (Kd = 0.065 nM, Bmax = 0.047 pmol/mg protein) or lower affinity sites (Kd = 0.49 nM, Bmax = 0.23 pmol/mg protein). Studies using membrane fractions of PEO14 cells indicated that this cell line has only a single lower affinity binding

site (Kd = 0.56 nM, Bmax = 0.31 pmol/mg protein). However, RT-PCR analysis
indicated the presence of mRNA from both ETA and **ETB** receptors
in PEO4 and PEO14 cell lines. Exogenous addition of ETs to PEO4 and PEO14
cells at concentrations of 10(-10)-10(-7)M resulted in specific
dose-dependent increases in cell number for ET-1 (with maximum effects at
10(-10) and 10(-9)M for PEO4 and PEO14, respectively) and ET-2 (maximum
effects at 10(-8) and 10(-9)M for PEO4 and PEO14, respectively) but not
for ET-3. Experiments on the growth of PEO14 cells using BQ123 (ETA-R)
antagonist and **"antisense"** oligonucleotide against the ETA-R, in
the absence of exogenous ETs, suggested that immunoreactive ET-1-like
material secreted by PEO14 cells can affect their growth in an autocrine
manner. These results would be consistent with ET-1 acting as a possible
autocrine growth regulator in human **ovarian** carcinoma cells.

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=> s schneider r?/au,in;s jamal s?/au,in

'IN' IS NOT A VALID FIELD CODE
L17       1256 FILE MEDLINE
L18       1615 FILE CAPLUS
L19       1409 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L20        880 FILE EMBASE
L21        844 FILE WPIDS
L22          8 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L23       6012 SCHNEIDER R?/AU,IN


'IN' IS NOT A VALID FIELD CODE
L24         47 FILE MEDLINE
L25         48 FILE CAPLUS
L26         77 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L27         45 FILE EMBASE
L28          1 FILE WPIDS
L29          7 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L30        225 JAMAL S?/AU,IN

=> s l23 and l30

L31          0 FILE MEDLINE
L32          1 FILE CAPLUS
L33          0 FILE BIOSIS
L34          0 FILE EMBASE
L35          1 FILE WPIDS
L36          0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L37          2 L23 AND L30

=> dup rem l37;d cbib abs
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PROCESSING COMPLETED FOR L37
L38 1 DUP REM L37 (1 DUPLICATE REMOVED)

L38 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 1
2000:790734 Document No. 133:329575 Cancer treatment with endothelin receptor antagonists. **Schneider, Robert J.**; Jamal, Sumayah (New York University, USA). PCT Int. Appl. WO 2000067024 A1 20001109, 64 pp.
DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA,
CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US11990 20000503.
PRIORITY: US 1999-305084 19990504.

AB The present invention relates to therapeutic protocols and pharmaceutical compns. designed to treat and prevent cancer. More specifically, the present invention relates to a novel method of treating cancer using antagonists to the endothelin B receptor (ETB) or inactive mimic forms of endothelin-1. The pharmaceutical compns. of the invention are capable of selectively inhibiting the early events assocd. with the development of cancer. The present invention further relates to screening assays to identify compds. which inhibit ETB activation.

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